NEURAL INJURY FOLLOWING TRAUMATIC ANTERIOR SHOULDER DISLOCATION

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To Candi, Luke and Zanthe

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TABLE OF CONTENTS

PART I : LITERATURE REVIEW

I.1. INCIDENCE AND TYPE OF LESION

There is no agreement in the literature and reports show a variable overall incidence of nerve injury following shoulder dislocation. Rowe reports 5.4%; De Palma, 5%; McLaughlin reports 2% in recurrent dislocations and 10% in acute dislocations; Watson-Jones (1936) reported 14% and Mumenthaler and Schliack, 15%. Pasila (1978) reports 21% and Brown reports 25%. In a very careful electromyographic study of 73 patients, Blom and Dahlback showed the incidence of nerve injury to be 35% and also showed that in the over 50 year age group the incidence rose to over 50%.

Probably the most important fact to be learned from this review of incidence is pointed out by Johnson and Bayley, where they stress that this is a complication usually of the older age group. They say that after acute anterior dislocation in the over 45 year group the most likely complications are nerve injury and rotator cuff tear. In patients younger than 45 the most likely complication is recurrence of the dislocation.

The incidence of isolated axillary nerve injury varies between 10% and 20% and can be expected to spontaneously recover up to one year following injury (Leffert 1965; Pasila 1980). Pasila (1980) reviewed 226 patients with acute dislocation of the shoulder and found 19 (8%) axillary nerve injuries and 25 (11%) brachial plexus injuries. Blom and Dahlback showed a remarkably high incidence of axillary nerve lesions following shoulder dislocation or fracture of the humeral neck. They studied 73 patients. 26 had nerve injuries of which 22 were

73 patients. 26 had nerve injuries of which 22 were confined to the axillary nerve. In their series the remaining 4 cases were axillary and musculocutaneous nerves, 1; axillary and radial nerves, 1; radial nerve, 1; and musculocutaneous and median nerves, 1. Another series that reported various combinations was that of Liveson. 11 patients with shoulder dislocation and nerve injury were reported. The axillary nerve was involved in all but one. Only in 3 was it isolated. The posterior cord was injured in 5 cases. The entire brachial plexus was involved in 2 patients. There was damage to the musculocutaneous nerve in 5 patients. This is not very common and only 3 cases prior to this have been reported in the literature (Blom and Dahlback/Seddon H. (1975)/Stevens)

There has been only one case of isolated suprascapular nerve lesion after dislocation reported (Zoltan). The only other time it is mentioned in passing is by Coene and Narakus who report 5 axillary nerve lesions with suprascapular nerve involvement but it is not clear in the paper whether those were directly from shoulder dislocation.

In Summary:

1. Overall incidence is from 5 - 35%
2. It is commonest in the elderly.
3. The commonest injury is the axillary nerve but any branch of the infra-clavicular brachial plexus may be involved.
4. The suprascapular nerve, the only

branch of supraclavicular brachial plexus to be involved, has only been reported once as an isolated injury. Its involvement has not been appreciated in the literature.

I.2. MECHANISM OF INJURY

The anatomical proximity of the infraclavicular brachial plexus, especially the axillary nerve to the glenohumeral joint, make it prone to injury during shoulder joint dislocation. By far the commonest mechanism of injury is local traction on the plexus (Milton (1953), Stevens, Gariepy). Much less commonly direct pressure on the nerves by the humeral head (Gariepy) or by a haematoma following vascular injury (Allende, Nash) may occur. Very uncommon is laceration of nerves by fracture fragments, in particular, the vertebral border of the scapula (Leffert and Seddon 1965).

According to Milton, McGregor postulated that the axillary nerve is crushed between the head of the humerus and the axillary border of the scapula. Most authors to date do not agree with this and the theory put forward by Stevens in 1934 still holds. He pointed out that the axillary nerve normally lies across the anterior surface of the subscapularis muscle. It comes off the posterior cord of the brachial plexus and then runs over the anterior surface of the subscapularis muscle. It then angulates sharply back posteriorly to travel along the inferior shoulder joint capsule. It leaves the axilla through the quadrangular space, below the lower border of the teres minor muscle, where it hooks around the posterior and lateral humerus on the deep surface of the deltoid muscle. Stevens makes the point that the head of the anterior dislocating humerus displaces the subscapularis tendon and

muscle forward and so creating traction and direct pressure on the nerve.

To date the most definitive work in the literature of possible mechanisms of nerve injury after shoulder dislocation is by GW Milton (1953). He used 15 cadavers to do an anatomical study of the possible mechanisms of axillary nerve palsy and tension created on the other branches of the brachial plexus during dislocation and reduction of the shoulder. The results and findings of this study are worth reiterating as they illustrate principles necessary to the understanding of this type of nerve lesion and clinical implications thereof.

In the first part of the study the shoulders were in the reduced position and the experiments were as follows:

With the arm in the anatomical position, all terminal branches were lax.

Manoeuvre no. 1: Strong downward traction applied to the adducted arm which was then rotated internally and externally to the fullest extent while maintaining traction. Findings were:

1. Downward traction produced traction on the plexus
2. Downward traction and internal rotation increased tension on the axillary and radial nerves.
3. Downward traction and external rotation caused a slight increase in radial nerve tension.

Manoeuvre no. 2: Lateral traction exerted with the

shoulder abducted to 90 degrees also in the extremes of rotation. Important findings were:

1. With the arm at 90 degress there was little traction in the posterior cord or its branches.

2. External rotation increased traction on the musculocutaneous nerve.

Manoeuvre no. 3: Traction and hyperabduction. This produced significant pull on the plexus.

In the second part of their study the shoulders were dislocated by hyperabduction and external rotation and then taken through the manoeuvres described above. The important findings were as follows:

The axillary nerve was tightly stretched across the humeral head during the hyperabduction stage of the dislocation.

Once the shoulder was dislocated, there was no tension on the plexus with the arm by the side.

Manoeuvre no. 1: Axillary nerve tension was increased by downward traction and internal rotation especially if the axillary nerve remained hooked over the head of the humerus rather than sliding down to its normal position at the humeral neck. If the arm was first externally rotated however, the nerve could slip down the head to the neck of the humerus and tension would be relieved.

Internal rotation with arm at the side increased tension of the axillary nerve, radial nerve and posterior cord.

Manoeuvre no. 2: With the shoulder abducted to 90 degress traction on the arm increased tension of all the branches of the brachial plexus except the axillary nerve.

Tension on the radial nerve was greatest in internal rotation, and the musculo-cutaneous nerve in external rotation.

Manoeuvre no. 3: In the hyperabducted shoulder, tension on the plexus was decreased although the axillary artery was stretched.

The most important message from these findings, apart from understanding the injury, is its application to reduction of dislocated shoulders.

1. When using the Hippocratic method of longitudinal traction, the shoulder should first be externally rotated to allow the axillary nerve to slip down off the head of the humerus before longitudinal traction is performed. The arm should be near as possible to the side where no tension on the plexus occurs. Traction in abduction should be avoided as this increases tension in all the branches of the plexus. The heel should be directed medially in the axilla and not upwards and/or laterally as this avoids direct pressure on the plexus.

2. I think the Kocher manoeuvre has the greatest chance of causing plexus damage and prefer to use the Hippocratic method. This is especially so if the shoulder is not reduced and the manoeuvre is completed. The posterior cord, axillary and radial nerves are at risk with the internal rotation manoeuvre.

I.3. DIAGNOSIS

The fact that the incidence of brachial plexus injury is anything from 5 - 35% following shoulder dislocation means that the diagnosis should first and foremost be made on a high index of suspicion. This should be even greater in the over 50 year age group where incidence is over 50% (Blom and Dahlback)

Obviously the most important diagnosis to make in the first instance is that of the shoulder dislocation. This will be clear from a history of trauma, clinical examination of the patient, and x-ray findings.

It is imperative before attempting reduction to examine for neurovascular deficit in the limb concerned. The neurological evaluation is made very difficult and may be obscured by (a) the presence of pain in the dislocated shoulder, (b) pain associated with any attempted movement and (c) immobilization in the early post-traumatic period. However there is no substitute for a thorough and meticulous clinical evaluation of the patient. Motor function in particular can be assessed by placing the hand on the muscle to be assessed. Attempt at movement can be felt as a contraction by the examiner without the patient moving the joint. This will suggest that the muscle's nerve supply is intact.

Blom and Dahlback have shown that the usual sensory testing of the axillary nerve (i.e. checking the skin sensation over the deltoid muscle laterally in region of the "Sergeant's stripes" is completely unreliable. They have

shown that the majority of patients with axillary nerve damage have no sensory loss, not even three patients who, by electromyography, showed complete denervation of the axillary nerve.

If the diagnosis of nerve injury is suspected clinically it can certainly be confirmed on electromyography (Liveson; Blom and Dahlback) within a month after injury. The best time for the diagnosis to be significant is after 3 weeks (Blom). Although this is the current thinking in the literature I do not believe that the EMG is of value. Firstly, I firmly believe that the diagnosis is a clinical one and EMG will only provide confirmation of this. Secondly from a practical point of view the surgeon wants to differentiate a neurapraxia and axonotmesis from nerve rupture. Again it has been shown that neurapraxia can be diagnosed at 3 weeks by EMG (Blom). By this time and certainly before 3 months there is also clinical improvement to confirm the clinical diagnosis of neurapraxia. So the important area where the surgeon requires information is differentiating axonotmesis from nerve rupture. Here the early EMG usually shows total denervation for both and therefore is of no help. It may start to show the beginnings of re-enervation by 2 - 3 months. This will signify axonotmesis and will encourage the surgeon not to explore. All too often then, this does not improve any further, leaving a functionless result. Too long an expectant delay and valuable time lost for exploration and repair leads to a less than satisfactory result.

In summary:

1. Older people (over 50 years) are particularly prone to nerve injury.
2. There is no substitute for a thorough clinical examination at the time of injury and at subsequent follow up.
3. Sensory testing of the axillary nerve is unreliable.
4. EMG may help to clinch the diagnosis but its clinical application to further management is doubtful.

I.4. PROGNOSIS

It is a well known fact that nerves have good elastic properties. This fact always fascinated me in severe trauma cases, like traumatic amputations following train accidents, where the only tissue that survived the crushing and stretching was the neural tissue connecting the amputated limb to the body. This observation combined with the fact that the infraclavicular brachial plexus is far removed from an anatomical point of anchorage (cervical spine) makes the plexus more extensible and less liable to serious damage. This explains why the majority of traction injuries of the brachial plexus following dislocation have a good prognosis.

In 1910 Delbet and Cauchoix published a study on plexus lesions accompanied by shoulder dislocation. They concluded that prognosis was favourable. This was reiterated by Stevens in 1934. In 1962 Gariepy, Derome and Laurin concluded that these lesions are always in continuity, prognosis was excellent and no surgical exploration is indicated. In 1965 Leffert and Seddon presented a series of 31 infraclavicular plexus palsies and most were seen after dislocation or fracture dislocation of the shoulder. They conclude that they are lesions in continuity and have a good prognosis. All but one patient had involvement of the deltoid in their series and in 50% of their cases. However, complete recovery of the deltoid was <u>not</u> obtained in all. Watson Jones (1952) reported 15 cases of axillary nerve palsy with shoulder dislocation. Two patients remained permanently paralysed. Therefore

many authors would have us believe that nerve injuries will recover and shoulder function will return to normal. A critical analysis of the literature reveals however that this is not so.

Another important fact to emerge from the study done by Pasila (1980) is that the rate of recovery of nerve lesions is significantly slower in patients over 50 years of age.

In Summary: The majority of lesions recover without surgery. The prognosis for recovery is therefore accepted as being excellent. However critically reviewing the literature shows many examples of cases with no return of function.

I.5. MANAGEMENT

There is general consensus in the literature that nerve injury following shoulder dislocation recovers non-operatively. Gariepy, Derome and Laurin (1962) stated that these lesions are always in continuity and that no exploration of the plexus is indicated. This was reiterated by Leffert and Seddon (1965) yet in 50% of their cases of axillary nerve palsy complete recovery of the deltoid was not obtained. In half of a series of patients with persistent post traumatic deltoid palsy, isolated or associated with palsy of other infraclavicular muscles reported by other authors, the deltoid failed to recover completely (Seddon 1947; Allende 1971; McManus 1976; Berry 1982). Watson-Jones (1952) reported 15 cases of axillary nerve palsy, 2 of which remained permanently paralysed. With the above results I cannot understand how operative treatment is not advocated in selected cases.

NON-OPERATIVE

Most reports in the literature are in agreement on the non operative management of these injuries. The principles are important and I feel should be applied to all patients regardless of whether they are to be treated operatively or non-operatively. These are:

1. Prevent stiffness and maintain mobility of joints.

2. Prevent swelling.

These aims are best realised by regular physiotherapy as well as occupational therapy. Combatting oedema by active

movement is important but often not possible with extensive paralysis. Elevation and passive movement of the limb is therefore imperative. Passive movement is also imperative to maintain a full range of joint mobility. Joint stiffness is especially important to prevent in the elderly where this is most likely to occur. In diffuse injuries a sling should also be supplied to support the shoulder and prevent subluxation of the gleno-humeral joint.

The importance of the above principles cannot be over-stressed because even if there is complete return of nerve and subsequent muscle function, if the joints are stiff the functional result will be very poor. Of utmost importance are those muscles that are far from the site of nerve injury, e.g. the intrinsics of the hand. Time to recover will be much longer for them than for muscles that are closer to the injury e.g. deltoid and therefore special attention should be given to all the joints of the hand.

There is no agreement on the use of regular galvanic stimulation of paralysed muscle to prevent atrophy. Although it is mentioned in the literature (Leffert 1965, 1985) it seems to have fallen out of favour and is not being used in most centres to-day. There is no evidence that a paralysed muscle with atrophy will not resume normal function once the muscle is re-innervated. However what does seem to be important is the time taken for re-innervation. It is generally accepted that the longer the delay before nerve repair is undertaken the worse the prognosis, until a time is reached when it is futile to consider repairing a nerve (Seddon 1972).

OPERATIVE

There are no papers in the literature dealing specifically with surgical repair of nerve injuries after shoulder dislocation. Narakas (1986), Sedel (1982) and Burge (1985), as a general principle, advocate exploration of infraclavicular nerve lesions and lesions of individual nerves around the shoulder.

In view of the following facts:

1. Most reports in the literature advocate non-operative treatment yet many report cases of poor nerve recovery.

2. The longer the delay before nerve repair, the worse the prognosis.

3. Much progress has taken place recently in anaesthesia and microsurgical techniques for nerve repair and grafting.

I hope to show conclusively in Part II of this dissertation that any nerve injury after dislocation that is not recovering progressively should be explored.

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PART II : BRACHIAL PLEXUS LESIONS CAUSED BY TRAUMATIC ANTERIOR DISLOCATION OF THE SHOULDER ; A REVIEW OF ALL PATIENTS SEEN IN THE HAND CLINIC AT GROOTE SCHUUR HOSPITAL OVER A 5-YEAR PERIOD (1980-1984)

II.1. ABSTRACT

In this study I reviewed 28 patients with brachial plexus lesions caused by shoulder dislocation. As far as can be established, this is the largest series reviewed in the literature to date.

Contrary to most other reports, the neurological lesions involved the supraclavicular as well as the infraclavicular brachial plexus. The only part of the supraclavicular brachial plexus affected was the suprascapular nerve, and this always recovered spontaneously. Isolated axillary nerve lesions were found to have the poorest prognosis for spontaneous nerve recovery. All lesions that showed no recovery after 3 - 5 months were explored and had either a graft or a neurolysis.

This study discusses the combinations of nerve lesions, their recovery and the indications for surgical intervention. I also suggest a classification perhaps more clinically relevant than the anatomical classification of Leffert and Seddon (1965).

II.2. INTRODUCTION

In 1910 Delbet and Couchoix published a classic study in which they pointed out that brachial plexus lesions resulting from shoulder dislocation involved the infraclavicular plexus and its terminal branches. They stated that prognosis for recovery of these lesions was favourable. This concept was reiterated by Stevens (1934).

In 1952 Watson-Jones reported 15 cases of shoulder dislocation complicated by axillary nerve palsy, two of them permanent.

Gariepy, Derome, and Laurin (1962) reported 6 cases of brachial plexus injury resulting from shoulder dislocation and concluded that these lesions are infraganglionic and **always** in continuity, the prognosis is excellent and no surgical exploration is indicated. This opinion was supported by Leffert and Seddon (1965) in their report on 31 cases of infraclavicular brachial plexus lesions of which 17 were associated with dislocated shoulder. Coene and Narakas (1986) reported 16 anterior shoulder dislocations, 5 with transient suprascapular nerve lesions.

These reports prompted a retrospective study of our patients as they did not correlate with our experience.

II.3. MATERIALS and METHODS

During the five years 1980 to 1984, 313 new patients with brachial plexus lesions were treated by Dr R.S. Boome at the Hand Unit at Groote Schuur Hospital, Cape Town. Of these, 28 were caused by shoulder dislocation. All were documented carefully on referral and at follow-up. This report is based on final evaluation by Dr J. Travlos who was not involved with their initial management.

The series consisted of 19 males and 9 females aged from 17 to 82 years and a mean of 42.6 years. 7 patients (1 female) were 17 to 28 years of age and 15 patients (5 females) were over the age of 40 years (Fig II.1). A simple fall was the most common cause of injury (Table II.1). All patients were referred for loss of function in the limb, the skeletal injuries having been already treated elsewhere. The commonest injury was a simple dislocation of the shoulder (Table II.2) and the severity of the skeletal injuries did not match the severity of the plexus injuries. There were no posterior dislocations. The interval between injury and first examination at the Brachial Plexus Clinic was under 1 month for 18 patients and the remainder before 10 months (mean 5.9 weeks) except 1 patient who was seen for the first time at 4 years. (Fig II.2)

Motor recovery was recorded as set out in the Medical Research Council Report on Peripheral Nerve Injuries (1954). Sensory Recovery was recorded as in Table II.3.

FIGURE II.1

Age distribution of 28 patients

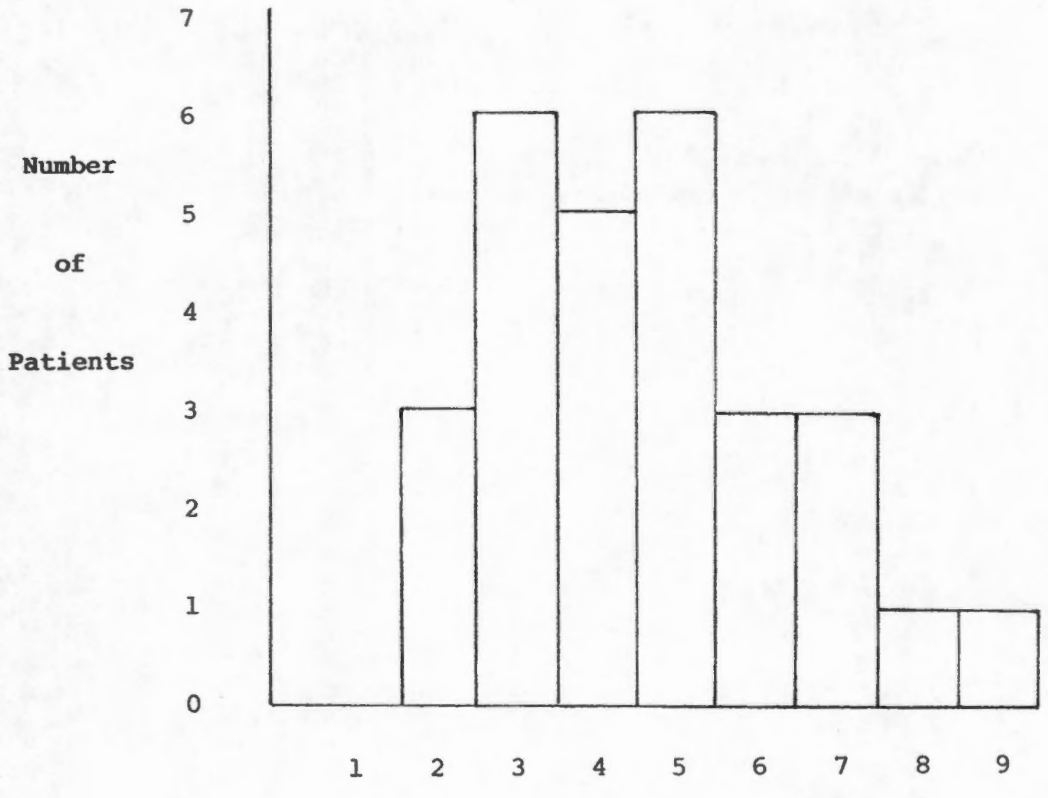

TABLE II.1 : Cause of injury

	Management	
	Conservative	Operative
Minor fall (level)	9	-
Major fall (height)	5	1
Motor vehicle/cycle	4	3
Direct blow to shoulder	3	-
Other	2	1

TABLE II.2 : Skeletal injury sustained

Dislocation only	21
Dislocation and fracture greater trochanter	2
Dislocation and fracture clavicle	1
Dislocation and fracture scapula	3
Dislocation shoulder, and fracture clavicle and scapula	1

FIGURE II.2

Interval between injury and first examination at our Centre

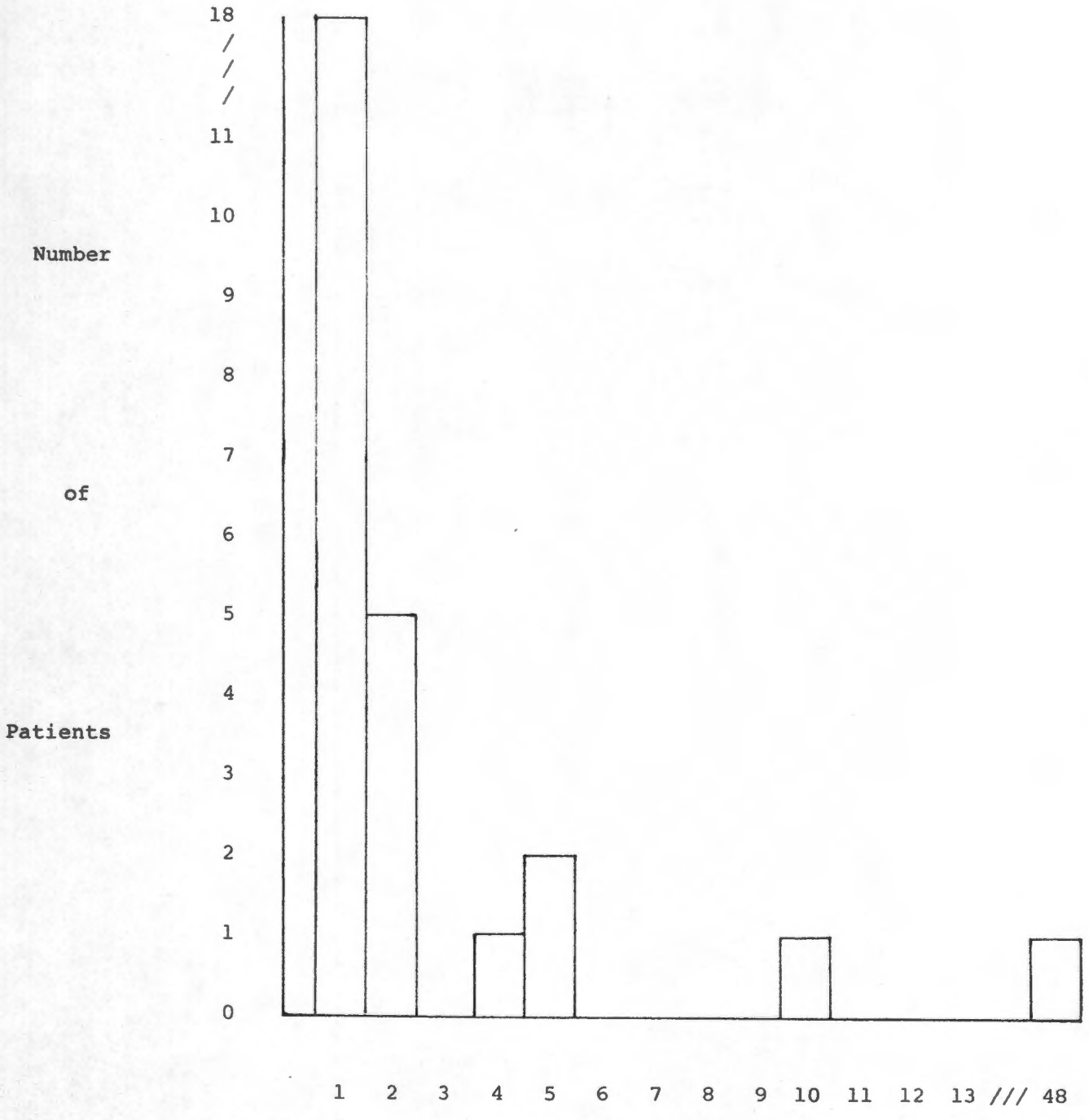

TABLE II.3 : Sensory recovery

Grade	
0	Nil
1	Deep pressure sense
2	Deep pressure sense Digit localisation
3	Gross paraesthesia Digit side localisation Gross stereognosis Minimal directional touch Two-point discrimination $<$10 mm
4	Mild paraesthesia
5	Normal

For the purpose of this study, the patients were divided into 2 treatment groups,

I NON-OPERATIVE MANAGEMENT (23 patients)

and II OPERATIVE MANAGEMENT (5 patients).

The 5 patients operated on were followed up for a minimum of 2 years post-operatively. The period of observation for those patients treated conservatively was one year for 8 patients, two years for 6 patients and three years for 9 patients. The decision to operate was based on no recovery by 3-5 months after injury with a persistent and unacceptable loss of function.

The operative technique for each depended on the cords and nerves most involved. The clavicle was divided when necessary.

All roots and nerves involved and not functioning fully were neurolysed using magnification and if there was no continuity sural nerve grafts were inserted. 4 neurolyses of affected nerves plus 3 sural nerve grafts of the axillary nerve were done in 5 patients. Axillary nerve grafts were done because the axillary nerve was found to be completely ruptured. These nerve grafts were 5-10 cms. long.

All patients had physical therapy for maintenance of joint range.

II.4. RESULTS

The lesions were classified anatomically by their infraclavicular involvement according to Leffert and Seddon (1965). These are:

A diffuse
B predominantly posterior cord
C predominantly lateral cord
and D predominantly medial cord.

MOTOR RECOVERY (See Tables II.4 - II.10)

There was no obvious difference in recovery of the different parts of deltoid (anterior, middle, and posterior). These were therefore grouped together. Wrist and finger flexion showed no significant difference in recovery time or strength, nor did wrist and finger extension, nor did median and ulnar intrinsics and these were therefore also grouped together.

1) **NON-OPERATIVE**

A : Diffuse infraclavicular : 10 patients

6/10 had involvement of the suprascapular nerve, all of which recovered fully.

At 14 months, all had recovered Grade 4 or 5 power of all arm and forearm muscle groups. However, 5 had poor functional results of the hand; 3 never regained any ulnar intrinsics and a further 2 had very stiff hands and fingers.

5/10 had stiff shoulders when last seen.

B : Posterior Cord : 10 patients

5 had isolated axillary nerve lesions and 5 combined lesions.

Deltoid recovery was poorest in the isolated axillary nerve lesions. 2 made no recovery. 1 attained Grade 4 power in the anterior fibres of deltoid but a stiff shoulder with poor abduction. 1 obtained full recovery by 4 months and 1 by 7 months.

Recovery of deltoid, triceps and forearm extensor muscles in the combined posterior group was excellent in that all 5 patients made a full functional recovery by 18 months, having shown early recovery at 6 months.

C : Lateral Cord : 4 patients

In all, biceps and brachialis recovered to Grade 5, with full elbow flexion.

D : Medial Cord : 6 patients

All recovered full motor hand function and at least Grade 4 power. One case was an isolated ulnar nerve lesion and one was an isolated median nerve lesion.

2) **OPERATIVE**

5 patients were operated on. 3 had diffuse infraclavicular lesions and 2 had isolated axillary nerve lesions.

3 of the 5 cases had axillary nerve grafts, using the sural nerve; 2 were for isolated axillary nerve lesions and 1 for

a diffuse lesion. None of these had any deltoid recovery at time of surgery which was 4-7 months post-injury. 1 of the 3 axillary nerve grafts (10cm long) was successful giving a good functional result. This was an isolated axillary nerve lesion.

4 cases had neurolyses. Of these, one was done because of no ulnar intrinsic recovery at 24 months and recovered from Grade 0 to Grade 3. The second case, a diffuse lesion, had neurolyses of the posterior and lateral cords 4 months post-injury and improved to full function.

One had a neurolysis of all cords 6 months post axillary artery repair and had no functional improvement at 20 months. He was left with a flail useless limb. The fourth case had a neurolysis of the axillary nerve graft 24 months after it failed, with only slight improvement.

SENSORY RECOVERY (see Table II.11)

Sensibilty of ulnar and median areas alone were considered because of their functional importance.

In the non-operative group there were 17 patients with sensory involvement of which 11 obtained complete recovery. 6 had adequate follow up but incomplete recovery. All however obtained a useful grade of sensibility i.e. Grade 3 or more.

In the operative group, only 2 patients had sensory involvement. 1 recovered fully and 1 had incomplete recovery.

TABLE II.4 : Diffuse infraclavicular lesion - motor recovery - NON OPERATED CASES

A = Initial examination
B = Final examination

Case No.	Follow up (months)	Deltoid A/B	Elbow Flexion A/B	Elbow Extension A/B	Wrist and Digit Extension A/B	Wrist and Digit Flexion Median & Ulnar A/B	Median and Ulnar Intrinsics A/B
1	12	0/4	0/4	0/4	0/4	0/4	0/3
2	5	4/5	4/5	2/4	1/4	1/4	1/3
3	30	0/5	0/5	0/5	0/5	0/5 *	0/5
4	12	4/5	0/5	0/5	0/5	0/5	0/0
5	9	0/4	0/4	0/4	0/4	0/3 *	0/Stiff
6	12	0/4	0/4 +	0/5	0/5	0/5	0/4
7	3	0/5	0/5	0/5	0/5	0/5	0/5
8	14	0/5	0/4	0/4	0/4	0/4	0/0
9	12	0/5	0/4	0/4	0/4	0/3	0/0
10	3	0/3 +	0/2	0/3 +	0/3 +	0/3 +	0/3 +

* Stiff hand

TABLE II.5 : The posterior cord - shoulder abduction - NON OPERATED CASES

Case no.	Nerve injury	Period of observation from time of injury (months)	Deltoid power (initial)	Deltoid power (final)	Functional result/remarks
11	Isolated axillary N	10	0	0	Operation planned - no further follow-up
12	Isolated axillary N	68	0	4 ant. fibres only	30° abduction stiff shoulder
13	Isolated axillary N	7	0	4	Full A ROM
14	Isolated axillary N	4	0	5	Full recovery
15	Isolated axillary	5	0	0	EMG showed poor renervation deltoid - operation planned: did not arrive for further follow-up
16	Axillary N combined	18	0	5	
17	Axillary N combined	14	0	5	
18	Post cord	3	0	5	
19	Post cord	12	0	4	
20	Post cord	12	4	5	Pain in shoulder

TABLE II.6 : **Posterior cord - recovery of extensor muscles** - NON OPERATED CASES

		Initial Examination			Final Examination		
Case no.	Period of observation from time of injury (months)	Elbow Extension	Wrist Extension	Extension of Digits	Elbow Extension	Wrist Extension	Extension of Digits
18	3	0	0	2	5	5	5
19	12	0	0	0	4	4	4
20	36	0	0	0	5	5	5

TABLE II.7 : Recovery of flexion of the elbow after lateral cord or musculocutaneous nerve injury - NON OPERATED CASES

Case no.	Period of observation from time of injury (months)	Associated Nerve Lesion	Biceps and Brachialis (initial)	Biceps and Brachialis (final)	Remarks
16	12	Axillary N	0	5	Full elbow flexion
17	14	Axillary N	0	5	Full elbow flexion
18	3	Posterior cord	3	5	Full elbow flexion
19	2	Posterior cord	3	4	Full elbow flexion

TABLE II.8 : Medial cord injury - motor recovery - NON OPERATED CASES

Case no.	Period of observation from time of injury (months)	Associated nerve injury	Initial Examination: Flexor carpi ulnaris	Initial Examination: Flexor digit profund (medial)	Initial Examination: Median intrin	Initial Examination: Ulnar intrin	Final Examination: Flexor carpi ulnaris	Final Examination: Flexor digit profund	Final Examination: Median intrin	Final Examination: Ulnar intrin
18	3	posterior cord	5	5	4	4	5	5	5	5
19	60	posterior cord	4	4	4	4	4	4	4	4
20	36	posterior cord	0	3	0	0	5	5	4	4
21	3	* mainly median nerve	FCR 2 +	FDP I 0	1	5	FCR 5	FDP I 5	5	5
22	2	mainly ulnar nerve	3	3	5	3	4	4	5	4
23	26	ulnar and median	5	3	3	3	5	5	5	5

FCR = Flexor carpi radialis

FDP I= Flexor digitorum profundus (Index)

TABLE II.9 : Diffuse infraclavicular lesion - motor recovery - OPERATED CASES

Case no.	Period of observation and operation performed	Deltoid	Elbow Flexion	Elbow Extension	Wrist Extension	Extension Digits	Wrist Flexion		Flexion Digits		Median Intrin	Ulnar Intrin
							Median	Ulnar	Median	Ulnar		
24		0	0	0	0	0	1	1	1	1	0	0
	24 months post injury	5	5	5	5	5	5	3	5	3	3	0
	Neurolysis medial cord at 24 months post injury				NO ULNAR INSTRINSIC RECOVERY							
	2 months and 24 months post operation	5	5	5	5	5	5	3	5	3	3	3
25		0	0	0	0	0	0	0	4	4	4	3
	4 months post inury	0	2	2	4	5	5	5	5	5	5	5
	Axillary nerve graft sural - 8 cm x 2 at 4 months				with neurolysis post and lateral cords							
	24 months post operation	0	5	3 +	5	5	5	5	5	5	5	5
28	Axillary artery repair	0	0	0	0	0	0	0	0	0	0	0
	6 months post axillary artery repair	0	0	0	0	0	0	0	2 +	2 +	2 +	2 +
	Neurolysis all cords at 6 months											
	26 months post neurolysis	0	0	0	2	2	2	2	3	3	3	3

TABLE II.10 : The posterior cord - shoulder abduction - OPERATED CASES

Case No.	Nerve Injury at examination	Period of observation (months)	Deltoid Power	Operation	Remarks
26	Isolated axillary nerve	Initial	0		
		Pre-op (7 months post injury)	0	Axillary nerve graft using sural nerve 10 cm x 2	Axillary nerve avulsed from post cord
		24 months post-op	4 +		
27	Isolated axillary nerve	Initial	0		
		Pre-op (7 months post injury)	0	<u>Operation 1</u> Axillary nerve graft using sural nerve 5 cm x 2	Axillary nerve avulsed from post cord
		24 months post axillary nerve graft	0	<u>Operation 2</u> Neurolysis axillary nerve	
		36 months post neurolysis	2		Very poor functional result

TABLE II.11 : Sensory recovery in the hand

Case no.	Period of observation from time of injury (months)		Initial Examination		Final Examination	
			Median	Ulnar	Median	Ulnar
	NON OPERATED					
1	12		2	2	5	5
2	5		4	3	5	3
3	30		0	0	5	5
4	12		2	2	4	3
5	9		2	2	5	5
6	2		0	0	4	4
7	2		4	4	5	5
8	14		1	1	5	5
9	12		1	1	4	4
10	3		0	0	4	4
11	-		*	*		
12	-		*	*		
13	-		*	*		
14	-		*	*		
15	-		*	*		
16	-		*	*		
17	2		4	5	5	5
18	3		5	4	5	5
19	18		4	3	5	5
20	36		3	3	5	4
21	3		3	*	5	*
22	2		*	4	*	5
23	26		*	4	*	5
	OPERATED					
24	24	Post-op	3	3	5	3
25	8		0	0	5	5
26			*	*		
27			*	*		
28			*	*		

* indicates no loss initially

II.5. DISCUSSION

The majority of the nerve injuries reviewed were referred from other centres. Therefore no conclusions can be drawn in this study about the general incidence of brachial plexus lesions following dislocation of the shoulder.

The classification used by Leffert and Seddon (1965) was found to be too broad and did not relate closely enough to the predominant loss of function or the functional recovery of lesions. It also did not take into account the suprascapular nerve involvement. I therefore suggest a more clinically applicable classification set out in Table II.12.

Since the traction on the brachial plexus is usually exerted laterally in shoulder dislocation at a point relatively far removed from an anatomical point of anchorage (spinal cord), the elasticity of the nerve roots protect them from severe damage. Most of the traction is borne by the infraclavicular plexus and the branch of this part that is nearest its anchorage is the axillary nerve, which explains its increased liability to injury (Leffert 1985).

Proximity to its anchorage may also be the reason for only the suprascapular nerve being involved in the rare supraclavicular plexus lesions.

TABLE II.12

Nerve injuries grouped by the predominant loss of function

1.	Diffuse infra- and supraclavicular nerve lesion	8
2.	Diffuse infraclavicular nerve lesion	7
3.	Isolated axillary nerve lesion	7
4.	Axillary nerve and lateral cord lesion	2
5.	Posterior cord and medial cord lesion	1
6.	Medial cord lesion	3

In assessing the time to recovery for deltoid it is of note that all those that recovered to Grade 4 or 5 did so by 18 months. They also showed at least Grade 1 power in the first 2 months after injury and all showed at least Grade 3 by 6 months. Recovery for deltoid was poorest in the isolated axillary nerve lesion. The signs of neurapraxia and neurotmesis are similar until the neurapraxia wears off and I feel there is no indication to explore before 3-5 months. Longer than this is disadvantageous as results of nerve repair after this period are not as good. I therefore recommend that the indications for exploration be:

a) failure to recover progressively

b) no recovery 3-5 months post injury

Dr Boome does not routinely use E.M.G. because it cannot differentiate axonotmesis from nerve rupture. He prefers to explore if the above indications are present.

I also recommend that when doing an axillary nerve graft it is obviously essential to resect all damaged nerve. This may mean combined anterior and posterior approaches to the plexus. This point was illustrated in my series by the fact that the longest graft gave the best result.

In assessing sensation as a indicator of recovery, no patient with complete absence of motor recovery had deep pressure sensation and the majority of those that recovered without operation had deep pressure sensation within 2 months after injury. It is therefore suggested that this is a good indicator of recovery potential in the combined lesion. I found that gross sensory recovery always occurred before motor recovery.

I reiterate the very important function of physiotherapy to maintain mobility until motor function returns.

I note, contrary to the literature to date, that suprascapular nerve injury may complicate shoulder dislocation. As an isolated injury it has been reported in one case only (Zoltan). Other authors (Liveson, Narakas et al) have mentioned it in association with other brachial plexus injuries. This loss of function is not a rotator cuff tear as pointed out by Ludin et al (1975), Pasila et al (1978) and Johnson (1981), because all patients recovered full supra- and infraspinatus muscle function.

II.6. SUMMARY

Any type of brachial plexus lesion may occur after shoulder dislocation. Most have a good functional result without surgery.

The poorest prognosis is found in isolated axillary nerve lesions and diffuse lesions where, despite physiotherapy, some joint stiffness occurs before full recovery. If there is no recovery 3-5 months after injury, this is an indication for exploration, which may have to be extensive as failure of recovery may be due to inadequate resection of the damaged nerve.

Deep pressure sensation where applicable, appears to be a good indicator of recovery potential.

A more clinically applicable classification is recommended.

Injury of the suprascapular nerve may occur (6/28 in this series) but always recovered fully.

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